receptor tyrosine kinase and thereby modulating the receptor tyrosine kinase activation by G-protein-mediated signal transduction.

--23. The method according to claim 22, wherein said receptor tyrosine kinase is epidermal growth factor receptor (EGFR).--

--24. The method according to claim 22, wherein said compound affecting an extracellular G protein or G protein coupled receptor initiated signal pathway affects (i) a proteinase cleaving a precursor of a ligand for the receptor tyrosine kinase or (ii) a precursor of a ligand for the receptor tyrosine kinase.--

--25. The method according to claim 24, wherein the compound affects the proteinase by directly stimulating or inhibiting proteinase activity.--

--26. The method according to claim 24, wherein said precursor of a ligand is a membrane associated molecule.--

--27. The method according to claim 26, wherein said precursor of a ligand for the receptor tyrosine kinase is proheparin-epidermal growth factor (proHB-EGF) and said receptor tyrosine kinase is EGFR.--

--28. The method according to claim 24, wherein said proteinase is a membrane-associated proteinase.--

--29. The method according to claim 24, wherein said proteinase is a metalloproteinase.--

--30. The method according to claim 29, wherein said metalloproteinase is a zinc-dependent proteinase.--

--31. The method according to claim 24, wherein said proteinase activity is inhibited by batimastat.--

--32. The method according to claim 22, wherein said compound affects a cell which is different from the cell containing the receptor tyrosine kinase.--

--33. The method according to claim 22, wherein said receptor tyrosine kinase is selected from the group consisting of EGFR, HER-2, HER-3, HER-4, TNF receptor 1, TNF receptor 2, CD 30 AND IL-6 receptor.

--34. The method according to claim 22, wherein said receptor tyrosine kinase is selected from the group consisting of EGFR and other members of the EGFR family.--